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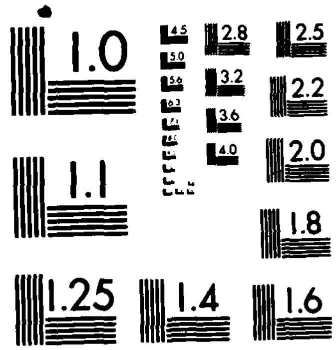

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heat can be accomplished during environmental stress at warm temperatures after atropine treatment.

Environmental Stress After Atropine Treatment

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Abstract

1. Atropine administration resulted in higher skin temperatures in both sensible and insensible environments and a higher core temperature in the hot environment, due to the reduction in whole body sweating.

2. The effect of heat storage (significantly higher after atropine) was shown to be greater in the hot environment due to inadequate sweat secretion for subsequent evaporative cooling. In the warm environment, enhanced skin blood flow resulted in more effective thermoregulation.

3. The results suggest that exercise in the heat can be accomplished during environmental stress at warm temperatures after atropine treatment.

Key words: Antimuscarinic drugs, Exercise, Heat stress, Sweating rate

INTRODUCTION

The evaporation of water from wet skin is dependent on air velocity, the vapor pressure gradient between the water on the skin and the water in the air, and the water vapor permeability of the clothing worn (Gagge and Nishi, 1976; Gonzalez et al., 1978; Nishi and Gagge, 1970). Atropine, which blocks cholinergic sweating, impacts on the volume of water which is secreted from sweat glands per unit area and this depresses evaporative heat loss (Berglund and Gonzalez, 1977; Craig and Cummings, 1965; Kolka et al., 1984). The evaporation of secreted sweat is the most important component of heat exchange between an individual and the environment when environmental temperature exceeds internal body and skin temperature. A high ambient water vapor pressure further limits evaporation from the skin due to an unfavorable skin to ambient saturation vapor pressure gradient in these humid environments (Gagge and Nishi, 1976; Nishi and Gagge, 1970). As long as the ambient dewpoint temperature does not exceed mean skin temperature, insensible heat loss will account for the most heat exchange in a humid environment.

In the present study, we evaluated heat exchange during low intensity exercise in two distinctly different environments with and without atropine injection. The two environments chosen were: one requiring primarily sensible or dry heat loss, and another necessitating evaporative heat loss for maintenance of body temperature.

METHODS

Subjects. Five heat-acclimated males volunteered for the study following consent procedures passed by the US Army Human Investigative Committee. The subjects had an average ($\pm$ SD) age of 22 $\pm$ 2 yr, weight of 77.1 $\pm$ 6.8 kg, height of 174.3 $\pm$ 1.6 cm, Du Bois surface area of 1.90 $\pm$ 0.07 m^2 and % body fat (hydrostatic weighing method) of 17.0 $\pm$ 4.0%.

Experimental Protocol. Testing occurred in a tropic-wind chamber in June. Subjects were fully oriented to all test procedures before testing was initiated. Subjects were tested in two environmental conditions; 42.3°C/20%rh (T_{dp} = 14.6°C), and 30.4°C/70%rh (T_{dp} = 23.8°C) in both a control experiment (saline injection) and after 2 mg of atropine sulfate was injected into the vastus lateralis. Subjects were injected with either atropine or saline 5 minutes before the initiation of each exercise-heat exposure. All five subjects walked at 1.34 $m \cdot s^{-1}$ which required a metabolic rate of ~350 W. The subjects were all in good health and had not taken any prescribed or unprescribed medication or alcohol during the course of the experiments. The exercise-heat exposures were randomized and were separated by two days.

Physiological Variables. Rectal (T_{re}) and mean skin temperature ($\bar{T}_{sk}$, 3 sites) and heart rate (HR) were continuously monitored (Kolka et al; 1984). Local dew-point temperature of the triceps area was measured by sensors strapped to the skin (Graichen et al., 1982) and local skin wettedness was calculated (Gonzalez et al., 1985) by:

$$\text{local } w = (P_{s,\,dpl} - P_w)/(P_{s,\,sk} - P_w) \qquad (1)$$

where $P_{s,\,dpl}$ is the saturated vapor pressure (kPa) of the local dewpoint sensor recording, P_w is the ambient water vapor pressure, (kPa) and $P_{s,\,sk}$ is the saturated vapor pressure at local skin temperature (kPa).

Metabolic heat production was calculated by open-circuit spirometry. Evaporative heat loss from the skin (E_{sk}, $W \cdot m^{-2}$) was calculated from body weight changes, before and after each exercise-heat exposure and corrected for convective (C_{res}) and evaporative (E_{res}) heat loss from the respiratory tract (Gagge, 1972). Subjects drank water ad libitum during all exposures.

Environmental Variables. Dry bulb (T_a), dew-point (T_{dp}, Eastern Instruments) and black globe (T_g) temperatures were monitored during the experiments. The wind speed was constant at 1.15 $m \cdot s^{-1}$. A series of experiments was conducted at a T_a of 42.3 $\pm$ 0.3°C and T_{dp} of 14.6 $\pm$ 0.1°C ("hot dry" environment). Another series of experiments was at a T_a of 30.4 $\pm$ 0.3°C and T_{dp} of 23.8 $\pm$ 0.5°C ("warm humid"). The average convective heat transfer coefficient (h_c) as calculated from Nishi and Gagge (1970) was 5.8 $W \cdot m^{-2} \cdot K^{-1}$ and the evaporative heat transfer coefficient (h_e) averaged 12.76 $W \cdot m^{-2} \cdot K^{-1}$. The radiant heat transfer coefficient (h_r) was 4.9 and 4.7 $W \cdot m^{-2} \cdot K^{-1}$ for the two conditions, respectively. The maximal evaporative capacity (E_{max}) of the environment was calculated (Gagge and Nishi, 1976) as 480 $W \cdot m^{-2}$ for the hot-dry environment after atropine and 355 $W \cdot m^{-2}$ for saline treated subjects. The E_{max} of the warm-humid condition was 270 $W \cdot m^{-2}$ and 201 $W \cdot m^{-2}$ for the atropine and saline treated subjects, respectively.

Heat balance was calculated incorporating environmental and physiological factors during the 30th minute of exercise for all four treatments (Kolka et al., 1984). This common point in time was chosen for statistical analysis following atropine treatment, since it is the last point in time that all necessary data for a complete thermal evaluation of the five subjects were obtained in all four treatments. The time correlates well with peak plasma levels of atropine (Berghem et al., 1980; Gosselin et al., 1960; Metcalf, 1981; Virtanen et al., 1982). The heat balance equation is:

$$S=M_{sk} - (\text{sensible heat loss})-(\text{skin evaporation}) \quad , W\cdot m^{-2} \tag{2}$$

$$\text{or } S=M_{sk} -h(T_{sk}-T_o)-wh_e (P_{s,sk}-P_w) \tag{2'}$$

where S is the rate of body heat storage ($W\cdot m^{-2}$); M_{sk} is the net heat flow determined from ($M-E_{res}-C_{res}$); h and h_e are combined radiative and convective heat transfer and evaporative heat transfer coefficients, respectively; w is the equivalent fraction of the total body surface (A_D) wet with sweat which was calculated from E_{sk}/E_{max}; $P_{s,\,sk}$ is the saturation vapor pressure (kPa) at mean skin temperature ($\bar{T}_{sk}$) and P_w is the ambient vapor pressure (kPa) calculated from the dew point.

Kolka et al., 1984 have described graphically on a psychrometric format the rate of heat storage (Gonzalez et al., 1978; Nishi et al., 1976: Nishi and Gagge, 1977) and that same format was utilized for the present study. Equation 2, rewritten in such a scheme, incorporates the heat balance equation as a function of ambient vapor pressure and dry bulb temperature gradient (Nishi and Gagge, 1977) in which

$$P_a - P_{s,sk} = (-\psi/w)\,[T_o - (T_{act} + \Delta\ T_{stor})) \quad , kPa \tag{3}$$

where; ψ is a constant which is the ratio of the transfer coeffients for sensible to insensible heat (h/h_e) with h_e incorporating the evaporative heat transfer constant ($16.5\ kPa\cdot K^{-1}\cdot h_c$). T_o is the operative temperature (Gonzalez et al., 1978) of the test chamber in which:

$$T_o = (h_r\ \bar{T}_r + h_C\ T_a)/(h_r + h_C) \quad , {}^oC \tag{4}$$

where; $\bar{T}_r$ (mean radiant temperature) was evaluated from $T_g + 2.2\sqrt{V}\,(T_g - T_a)$, in which V is the air velocity ($m\cdot s^{-1}$).

The temperature (T_{act}) in eg. 3 assumes each of the subjects is in thermal balance (S=0) regulated by sensible heat loss.

$$T_{act} = \bar{T}_{sk} - M_{sk}/h \quad , {}^oC \tag{5}$$

Any displacement in heat storage (ΔT_{stor}) during a given exercise transient may be calculated as:

$$\Delta T_{stor} = (\Delta T_{re}/\Delta t \cdot 0.97 \cdot m_b/A_D)/h \quad ,^oC \qquad (6)$$

where; $\Delta T_{re}/\Delta t$ is the change in rectal temperature per h, 0.97 is the specific heat content of the tissues ($W \cdot h^{-1} \cdot kg^{-1} \cdot {}^oC^{-1}$), m_b is the lean body mass (kg), A_D is the DuBois surface area (m^2) and h is the combined heat transfer coefficient ($W \cdot m^{-2} \cdot K^{-1}$). Equation 3 now is represented on psychrometric chart by a series of lines passing through a common point (CP = T_{act}, $P_{s,sk}$). Any effect of internal body heating causes a displacement of the CP with coordinates (T_{act} + ΔT_{stor}, $P_{s,sk}$). The rate of displacement becomes proportional to any combined body heating effects of atropine and the environment. The locus of any environmental condition (T_o, P_a) is represented by OP. If the CP and OP are connected, the point where this line crosses the 50% rh curve on a psychrometric chart represents an effective temperature (ET*) defined by T_o on the X axis of the chart. This ET* (Nishi et al., 1976; Nishi and Gagge, 1977) is a theoretical temperature of an isothermal enclosure in which a person exchanges the same total heat by (R + C) and evaporative loss from the skin. The ET*, however, can be used to integrate combined effects of T_o, P_a, physiological strain, and changes in heat balance caused by the atropine.

Statistical Treatment. A two way analysis of variance was utilized for all data at the time of peak drug effects (30 minutes of exercise) when a complete heat balance could be determined (Kolka et al., 1984). Linear regression coefficients were calculated for changes in heat storage over time. Data in the RESULTS are presented as mean $\pm$ SD. All differences reported are significant at $p < 0.05$.

RESULTS

All five subjects completed 100 minutes of exercise in the saline experiments and in the warm atropine condition. The average exposure in the hot environment after atropine injection was 71.6 $\pm$ 23.9 minutes, significantly ($p < 0.05$) less than the exposure time for the three other conditions.

Table 1 shows mean $\pm$ SD environmental and physiological variables at the 30th minute of exercise (38 minutes post drug injection) in the four conditions. Arm wettedness as measured by the dewpoint of the skin (Gonzalez et al.,1985) is graphically shown in Figure 1 for one subject. There was a significantly higher skin wettedness in both control experiments compared to atropine exposures, and furthermore, measured wettedness (arm) was generally higher in the warm environment than the hot environment for both treatments (Figure 1). Whole body skin wettedness, (Table 1) was depressed 60% in the hot environment and 70% in the humid environment after atropine treatment. This depression in wettedness and subsequent evaporation resulted in higher core (0.47°C) and skin (3.98°C) temperatures in the hot environment and a higher skin (2.44°C) temperature in the humid environment with no change in the core temperature. Metabolic heat production was not different between environments or after atropine injections.

The lowered evaporative heat loss combined with an unfavorable gradient ($\bar{T}_{sk}-T_a$) for sensible heat loss accounted for about a 300% increase in the rate of heat storage in the hot-dry environment after atropine. The rate of heat storage was increased by only 90% in the warm-humid environment after atropine due to the favorable gradient allowing sensible heat exchange (~5.0°C).

Table 2 gives some physiological properties essential to develop the psychrometric chart shown in Figure 2A and 2B for the saline and atropine exposures, respectively. This figure demonstrates the higher ET* present in both the hot and humid conditions after atropine injection compared to saline. A heightened physiological strain occurred in the individuals in the hot-dry environment compared to the humid exposure given the drug. The effect of ΔT_{stor} is shown to be greatest in the hot-dry condition with atropine (Table I, Figure 2A, 2B).

DISCUSSION

We evaluated heat exchange in two environments during low intensity exercise, with and without atropine treatment. The environments were chosen to require either evaporative or dry heat loss respectively, and were evaluated initially on the basis of having similar Wet Bulb Globe Temperatures (WBGT), a common emperical index to express environmental heat stress.

The suppressing effect of atropine on thermoregulatory sweating with subsequent body heating in the hot, dry environment was consistent with numerous studies (Craig and Cummings, 1965; Craig et al; 1969; Kolka et al; 1984; Robinson, 1953). The suppression of sweat secretion was of a similar magnitude (-45%) in the humid environment without significantly impacting on body cooling by dry heat exchange. For the same atropine dosage, the heart rate responses were markedly different in the two distinct environments of this study. An average 62% (58 $b \cdot min^{-1}$) elevation in heart rate occurred in the hot environment and a 42% (39 $b \cdot min^{-1}$) increase in the humid exposures. The cardiovascular effect of atropine is manifested primarily via vagal inhibition resulting in a release of parasympathetic inhibition with consonant increased cardiac frequency. This higher cardiac frequency and cardiac stress

presumably relates to the elevated shell temperature with accompanying cardiovascular stress, specifically, heightened venous pooling with resultant shifts in the blood volume, decreasing end disastolic volume, thus requiring increased cardiac frequency to maintain cardiac output.

Gonzalez et al., 1985 calculated sensible (dry) heat loss after atropine injection and demonstrated a greater dry heat loss with the decreased skin wettedness after atropine injection. This enhanced dry heat loss apparently parallels enhanced blood flow through this cutaneous site (arm). It is not apparent from our investigation whether the enhanced blood flow and increased heat loss is a local effect or a centrally mediated response to elevated core temperature, or both.

The ET* developed in this study increased in the hot environment after atropine, but was not altered by atropine in the less stressful dry but more humid condition as shown in Tables 1 and 2. Body temperature was effectively regulated even with a similar depression in whole body wettedness, due to the sensible heat loss that could occur in this environment. The negative values in Table 2 show an increase in relative dry heat gain by the body (represented by the arm in this case). More heat was gained from the environment in the control experiments as a result of the larger gradient for heat gain ($T_a \geq T_{sk} \cong 8^{o}C$). However, evaporation of secreted sweat was sufficient to regulate body temperature in the control experiments. Atropine administration resulted in a consistent depression of sweat secretion with far greater thermoregulatory consequence in the hot environment. Less heat was gained from the environment ($T_a \geq T_{sk} \cong 4^{o}C$), however, depressed evaporative heat loss resulted in greater heat storage and diminished ability to perform in this condition.

The Effective Temperature (ET*) used here allows a summing effect of environmental heat stress. This index can also be used in the heat balance equation. The ET* was 2.4°C lower in the warm-humid than hot-dry environment after atropine and was only 0.7°C higher (Figures 2A, 2B) than that apparent in the control (saline) warm-humid exposures. This lowered effective thermal stress may be associated with a compensatory cutaneous heat loss which was possible in the humid condition due to the favorable gradient for heat exchange between the body and the ambient air.

In conclusion, the effect of atropine on heat balance (primarily heat storage properties) resulted in an increased ET* of ~5.0°C in the hot-dry environment but only ~2.0°C in the less stressful dry but more humid environment, demonstrating the severity of the physiological strain after atropine with dry heat stress coupled with exercise.

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The views, opinions and/or findings contained in this report are those of the authors and should not be construed as official Department of the Army position, policy or decision, unless so designated by other official documentation. Human subjects participated in these studies after giving their informed voluntary consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 on use of Volunteers in Research.

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Figure Legends

Figure 1. Direct measurement of local skin wettedness for one subject of the upper arm during both saline and atropine treatments in the hot-dry and the warm-humid environments.

Figure 2 A. ET* loci in atropine experiments; T_{stor}, heat storage; P_{sk}, skin saturation vapor pressure; $T_{act} = \bar{T}_{sk} - M_{sk}/h$; OP, environmental conditions (operative temperature and ambient vapor pressure); CP, common point (T_{act} + T_{stor}, P_{sk}). B. ET* loci in saline experiments.

Table Headings

Table 1. Mean (± S.D.) 35 minute data for 5 males in control and atropine experiments. Wind speed, 1.15 $m \cdot s^{-1}$, Treadmill speed, 1.34 $m \cdot s^{-1}$; T_a = 42.3, T_{dp} = 14.6°C; T_a = 30.4°C, T_{dp} = 23.8°C.

Table 2. Physiological properties used for graphic representation of heat storage.

Table 1. Mean (± SD) 35 min[illegible] data for 5 males in control and atropine experiments. Wind speed, 1.15 $m \cdot s^{-1}$; Treadmill speed 1.34 $m \cdot s^{-1}$; T_a = 42.3°C, T_{dp} = 14.6°C; T_a = 30.4 °C, T_{dp} = 23.8°C.

	T_{re} (°C)	$\bar{T}_{sk}$ [illegible]	w (%)	M ($W \cdot m^{-2}$)	E_{sk} ($W \cdot m^{-2}$)	HR ($b \cdot min^{-1}$)	$P_{s,sk}$ (kPa)	E_{res} ($W \cdot m^{-2}$)	C_{res} ($W \cdot m^{-2}$)	ΔT_{stor} (°C)
Atropine										
Hot	37.69*	[illegible]*	.29*	203	139*	152*	6.70*	14.7	-2.4	10.9*
	(0.28)	(0.72)		(12)	(35)	(11)	0.63	(0.1)	(0.1)	(1.8)
Humid	37.14††	[illegible]*††	.27*	187	74*††	132*††	5.76*††	9.5	0.9	5.2*††
	(0.23)	(0.5[illegible])		(13)	(12)	(11)	0.58	(0.7)	(0.1)	(0.9)
Control										
Hot	37.24	[illegible]	.72	194	257	94	5.39	14.0	-2.3	2.5
	(0.12)	[illegible]2)		(18)	(19)	(7)	0.65	(1.3)	(0.2)	(0.9
Humid	37.15	[illegible]	.88	186	145	93	5.03	9.4	0.9	2.8
	(0.19)	[illegible]		(7)	(19)	(7)	0.55	(0.4)	(0.1)	(1.5)

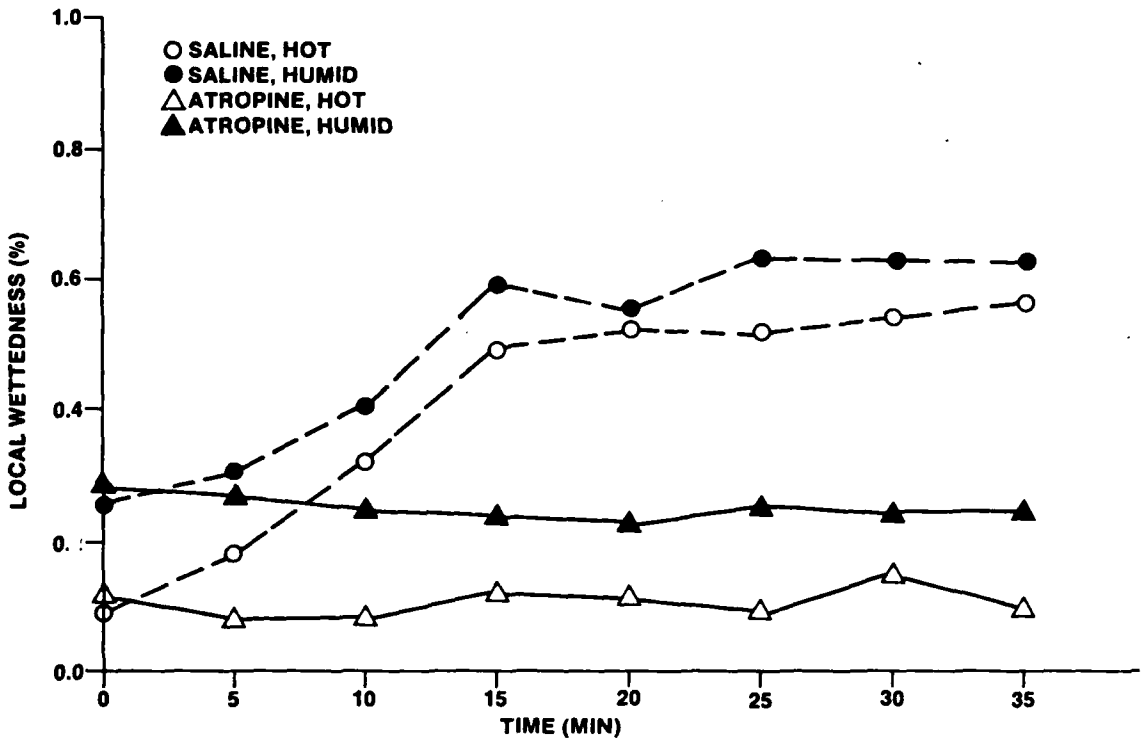
* Different from control $p < 0.$[illegible]
†† Different from Hot $p < 0.05$

w, total body surface wet wit[illegible]; M, metabolic heat production; E_{sk}, evaporative heat loss from the skin; $P_{s,sk}$ saturation vapor pressure at $\bar{T}_{sk}$; E_{res}, evaporative heat loss fro[illegible]espiratory tract; C_{res}, convective heat loss fro[illegible] the respiratory tract; ΔT_{stor}, change in heat storage.

Table 2. Physiological properties for graphic representation of heat storage.

	T_g (°C)	ET* (°C)	T_o (°C)	T_{act} (°C)	h ($W \cdot m^{-2} \cdot °C^{-1}$)	M_{sk} ($W \cdot m^{-2}$)	$P_{s,sk}$ (kPa)	CP ($T_{act}, P_{s,sk}$)	R + C ($W \cdot m^{-2}$)
Atropine									
Hot	41.4	37.7	40.5	20.5	10.8	191	6.7	20.5, 6.7	-24.3
Humid	31.6	35.3	32.2	18.7	10.5	176	5.8	18.7, 5.8	34.1
Control									
Hot	41.1	34.7	40.5	17.2	10.7	182	5.4	17.2, 5.4	-66.9
Humid	31.6	34.6	32.2	16.1	10.5	176	5.0	16.1, 5.0	8.5

Values are means ± SD. Atropine and control experiments in hot and humid conditions. T_g, globe temperature; ET*, effective temperature; T_o, operative temperature; $T_{act} = T_{sk} - M_{sk}/h$; h, heat transfer coefficient; M_{sk}, net heat flow; $P_{s,sk}$, saturation vapor pressure at T_{sk}; CP, common point coordinates; R+C, sensible heat loss.


1.0
0.8
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LOCAL WETTEDNESS (%)
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ATROPINE, HUMID
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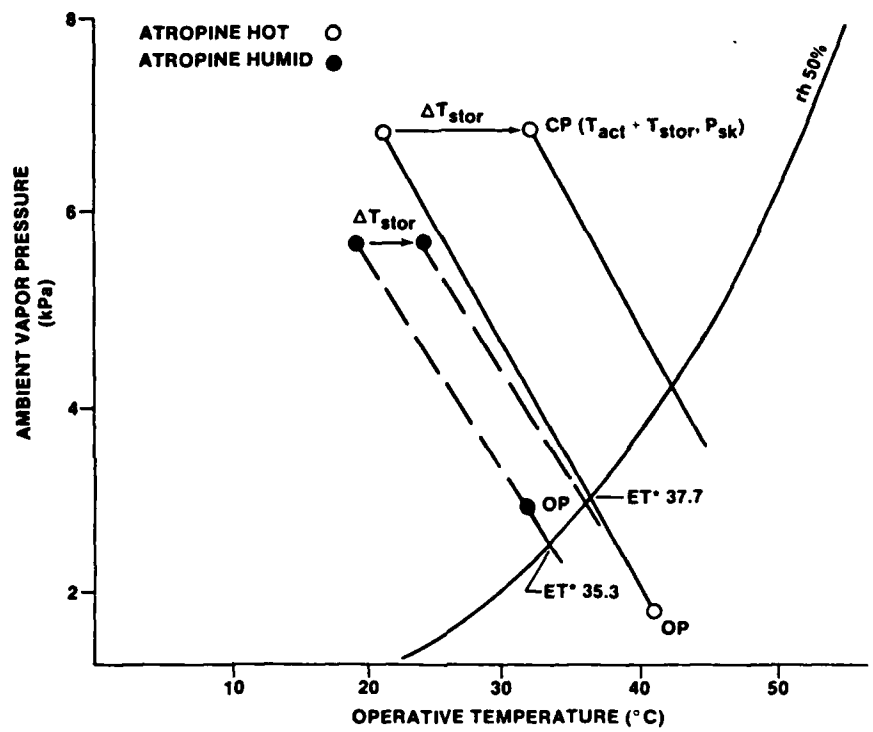
TIME (MIN)


ATROPINE HOT
ATROPINE HUMID
ΔTstor
CP (Tact + Tstor, Psk)
rh 50%
ΔTstor
OP
ET* 37.7
ET* 35.3
OP
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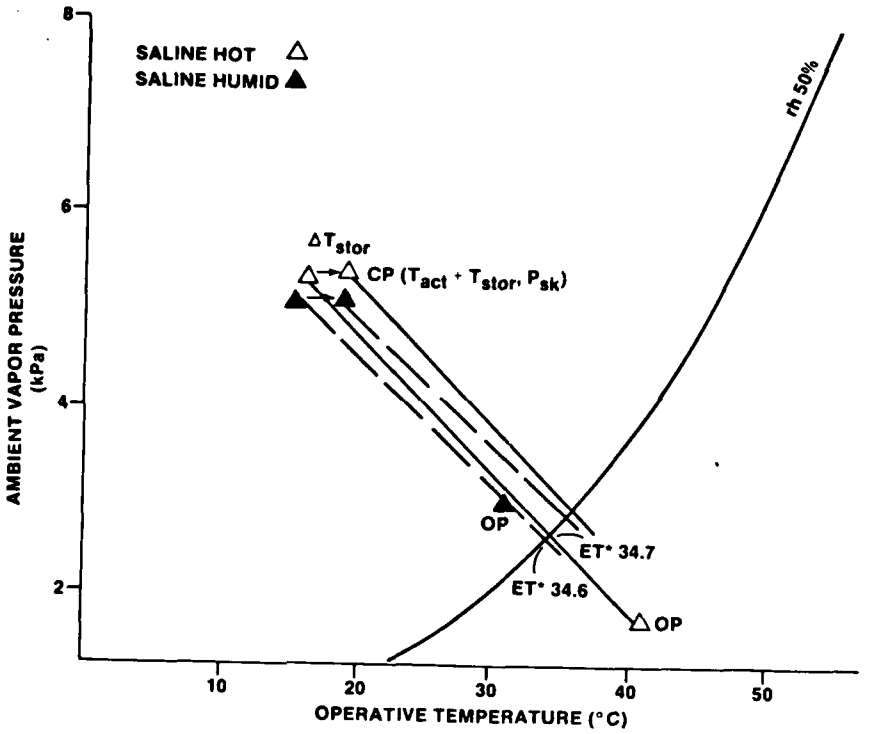
OPERATIVE TEMPERATURE (°C)


SALINE HOT △
SALINE HUMID ▲
rh 50%
ΔTstor
CP (Tact + Tstor, Psk)
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ET* 34.7
ET* 34.6
OP
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OPERATIVE TEMPERATURE (°C)

Table 1. Mean (± SD) 35 min ... data for 5 males in control and atropine experiments. Wind speed, 1.15 $m \cdot s^{-1}$; Treadmill speed 1.34 $m \cdot s^{-1}$, 42.3°C, T_{dp} = 14.6°C; T_a = 30 ... T_{dp} = 23.8°C.

	T_{re} (°C)	$\bar{T}_{sk}$ (°C)	w (%)	M ($W \cdot m^{-2}$)	E_{sk} ($W \cdot m^{-2}$)	HR ($b \cdot min^{-1}$)	$P_{s,sk}$ (kPa)	E_{res} ($W \cdot m^{-2}$)	C_{res} ($W \cdot m^{-2}$)	Δ ... (...
Atropine										
Hot	37.69*	...*	.29*	203	139*	152*	6.70*	14.7	-2.4	1...
	(0.28)	(...)		(12)	(35)	(11)	0.63	(0.1)	(0.1)	(1...
Humid	37.14††	...*††	.27*	187	74*††	132*††	5.76*††	9.5	0.9	5...
	(0.23)	(...)		(13)	(12)	(11)	0.58	(0.7)	(0.1)	(0...
Control										
Hot	37.24	...	.72	194	257	94	5.39	14.0	-2.3	2...
	(0.12)	...		(18)	(19)	(7)	0.65	(1.3)	(0.2)	(0...
Humid	37.15	...	.88	186	145	93	5.03	9.4	0.9	2...
	(0.19)	...		(7)	(19)	(7)	0.55	(0.4)	(0.1)	(1...

* Different from control $p < 0$...
†† Different from Hot $p < 0.05$

w, total body surface wet wit... ; M, metabolic heat production; E_{sk}, evaporative heat loss from the skin; $P_{s,sk}$ saturation vapor pressure E_{res}, evaporative heat loss fro... espiratory tract; C_{res}, convective heat loss from the respiratory tract; ΔT_{stor}, change in heat storage.

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